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ATP reduces the entry of calcium ions into the nerve ending by Blocking L-type calcium channels

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Abstract

© 2018 Park-media, Ltd. At neuromuscular junctions, ATP inhibits both the evoked and spontaneous acetylcholine release and inward calcium current operating via presynaptic P2Y receptors. It was shown in experiments with the frog neuromuscular synapse using specific calcium-sensitive dye Oregon Green Bapta 1 that exogenous ATP reduces the amplitude of calcium transient, which reflects the changes in the entry of calcium ions in response to the nerve pulse. The depressing effect of ATP on the transient was prevented by suramin, the blocker of P2 receptors. Nitrendipine, a specific blocker of L-type calcium channels, per se decreased the calcium transient amplitude and significantly attenuated the effect of ATP on the calcium signal. Contrariwise, the preliminary application of ATP to the neuromuscular junction completely eliminated the depressing effect of nitrendipine on the calcium response. The obtained data suggest that an essential component in the inhibitory action of ATP on the calcium transient amplitude is provided by reduction of the entry of calcium ions into a frog nerve ending via L-type voltage-gated calcium channels.

Keywords

ATP, Calcium channels, Calcium transient, Neuromuscular junction

References

[1] Sokolova E., Grishin S., Shakirzyanova A., Talantova M., Giniatullin R.//Eur. J. Neurosci. 2003. V. 18. P. 1254-1264

[2] Grishin S., Shakirzyanova A., Giniatullin A., Afzalov R., Giniatullin R.//Eur. J. Neurosci. 2005. V. 21. P. 1271-1279

[3] Khaziev E., Golovyahina A., Bukharaeva E., Nikolsky E., Samigullin D.//BioNanoSci. 2017. V. 7. P. 254-257

[4] Tsentsevitsky A.N., Samigullin D.V., Nurullin L.F., Khaziev E.F., Nikolsky E.E., Bukharaeva E.A.//Frogs: genetic diversity, neural development and ecological implications/Ed. Lambert H. New York: NOVA Pupl., 2014. P. 179-194

[5] Khaziev E., Samigullin D., Zhilyakov N., Fatikhov N., Bukharaeva E., Verkhratsky A., Nikolsky E.//Front. Physiol. 2016. V.7. P. 621. doi: 10.3389/fphys.2016.00621. eCollection 2016

[6] Robitaille R.//J. Neurosci. 1995. V. 15. P. 7121-7131

[7] Yamamoto T., Habuchi Y., Nishio M., Morikawa J., Tanaka H.//Cardiovascular Res. 1999. V. 41. P. 166-174

[8] Samigullin D.V., Khaziev E.F., Zhilyakov N.V., Bukharaeva E.A., Nikolsky E.E.//J. Vis. Exp. 2017. V. 125. P. 55122

[9] Tsentsevitsky A., Nikolsky E., Giniatullin R., Bukharaeva E.//Neuroscience. 2011. V. 189. P. 93-99

[10] Emmick J.T., Kwon S., Bidasee K.R., Besch K.T., Besch H.R. Jr//J. Pharmacol. Exp. Ther. 1994. 269. P. 717-724

[11] Hill A.P., Kingston O., Sitsapesan R.//Mol. Pharmacol. 2004. V. 65. No 5. P. 1258-1268

[12] Sugiura Y., Ko C.P.//Neuroreport. 2000. V. 11. No 13. P. 3017-3021